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Status epilepticus in children

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Aetiology and outcome of status epilepticus in children are different in comparison with adult patients. The main characteristics of status epilepticus in 112 children (age 6 months–15 years) are presented, with special attention to age, duration of status epilepticus, causes, medical complications and therapy. The greater part of these children was known to have had prior epilepsy, a considerable number with mental retardation.

Outcome in convulsive status epilepticus is influenced by cause, duration, age, the occurrence of medical complications and quality of treatment. Outcome in nonconvulsive status epilepticus is good and does not seem to be influenced by the treatment strategy. The use of a therapy protocol may prevent unnecessary delay and contribute to a better outcome.

Key words: status epilepticus children; causes; complications; therapy; outcome.

INTRODUCTION

This study was prompted by the experience that, in the different hospitals where the author worked, rather divergent opinions about the care for status epilepticus were encountered. Furthermore, status epilepticus was generally considered a frightening condition implying an important association with mortality or at least morbidity, which did not seem to be corroborated in actual practice.

First, the literature was reviewed, next, 112 cases of children admitted in a number of Dutch hospitals or treated in Special Centres for Epilepsy were collected and studied with respect to population characteristics, type of status epilepticus, causes, duration of the status, medical complications and strategy of therapeutic measures.

LITERATURE

Status epilepticus (SE) is a major neurological and medical emergency requiring treatment to prevent significant brain damage and possible mortality. SE is a seizure with a minimum duration of 30 minutes; any seizure type may extend to SE. A succession of generalized convulsive seizures without regaining consciousness is also considered SE[1]. The limit of 30 minutes is not a random choice; beyond that period various pathophysiological changes occur, starting in the substantia nigra[2,3]. These pathophysiological changes occur mainly in generalized convulsive SE (GC–SE) and are important with regard to outcome. Because of various unique features of SE in the neonatal brain we discuss SE only in children beyond the neonatal period.

In the general population both the very young and the elderly represent the populations most at risk of developing SE. The number of cases have a slight peak at <1 year of age and gradually decline until early adulthood[4]. Mortality in children is lower (2–3%) than in adult patients (25%); mortality increases with age and depends on the underlying cause[4]. The causes of SE are divided into idiopathic, remote symptomatic, febrile, acute symptomatic and progressive encephalopathy. Children without prior epilepsy are relatively younger, show a longer seizure duration and a longer postictal coma. SE is the initial presentation of epilepsy in 8–12% and occurs in 10–20% during the course of epilepsy, especially in cases of symptomatic epilepsy. Recurrent SE occurs mainly in children with an underlying neurological abnormality[5].

The risk of epilepsy following SE is increased in acute and remote symptomatic cases, but not in

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idiopathic SE[6]. The classification of SE follows the classification of seizures[7]. In children, excluding neonates, generalized convulsive SE is the most frequent occurring type.

Generalized convulsive status epilepticus (GC–SE)

Tonic–clonic and clonic (hemi-clonic) SE are the most prevalent types[8–10]. More than 75% of the children with convulsive SE has an age of three years or less[8,11,12]. The greater part (50–70%) had never had seizures before (Table 1).

The acute symptomatic group consists of central nervous system infections, metabolic problems, anoxia and head trauma[4,8–10,17]. The outcome of convulsive SE is especially determined by its cause. Outcome is worse in the acute symptomatic group in comparison with the idiopathic group or in patients with a chronic encephalopathy. Other factors of importance are age[8,9] and duration of SE[4,8,13,14].

A significant contribution of duration with respect to outcome of SE was noted only in the acute symptomatic group[16].

A relationship between cause and mortality has been found especially in cases caused by tumors, but also by anoxia and metabolic problems[4].

Fever alone, not associated with central nervous system infections was not associated with significant morbidity or mortality.

Generalized nonconvulsive status epilepticus (GNSE)

Generalized nonconvulsive SE include typical absence SE, atypical absence SE, encephalopathy related to electrical status epilepticus during sleep (ESES) and Landau–Kleffner syndrome (LKS).

GNSE may present severe diagnostic problems, especially in mentally retarded patients. The clinical presentation of GNSE in mentally retarded children is diverse and sometimes very subtle. An essential factor is a change in behaviour and/or awareness, in combination with ataxia, dysarthria, drolling, myoclonic jerks or twitching and sleep disturbances. EEG investigation is necessary and may show several epileptic EEG patterns: multifocal spikes and spike-waves, diffuse spike and slow waves or polyspike and slow waves.

In mentally retarded patients with epilepsy, e.g. children with West or Lennox–Gastaut syndrome, atypical absence SE is the most frequently occurring type of GNSE. Other types of SE in these children are tonic, tonic–clonic and myoclonic SE.

Typical absence SE is relatively rare in children. Most are already known to suffer from idiopathic generalized epilepsy with absences and/or generalized tonic–clonic seizures, without any neurological impairment. Clinically, prolonged episodes are seen with a disturbance of mental function in association with a continuous repetitive or intermittent spike and wave pattern on the EEG. The typical 3 Hz pattern may be present, but other more irregular patterns are no exception. Most patients are slow, drowsy, show some automatisms or myoclonic jerks. Consciousness may vary from a slightly delayed response to complete lethargy.

Benzodiazepines are successful for most cases. Precipitating factors include infections (mostly respiratory), medication problems and stress. Duration does not exceed 24 hours in most cases. Cognitive deterioration after typical absence SE in children has not been established.

Apart from complex partial SE, generalized NSE should also be distinguished from an acute confusional state caused by fever, trauma or metabolic problems[18], and from prolonged postictal encephalopathy[19].

Table 1: General characteristics of SE in children

Reference	8	12	10	13	14	15	16	This study
Number	239	67	84	52	97	193	193	112
Duration of SE (min)	>60	>60	>30	>30	>30	>30	>30	>30
Age (yrs)	<15	<15	<13	<18	<18	<14	<18	<15
Prior Epilepsy (%)	23	83	16	35	49	29	32	66
Causes (%)								
Acute Symptomatic	26	16	42	30	15	43	23	25
Chronic Encephalopathy	21	—	45	36	57	10	29	53
Idiopathic	52	—	13	32	28	46	48	22
Fever	28	—	6	21	16	32	24	4.5
Mortality (%)	11	3	6	6	7	6	3	11.5
Morbidity (%)	57	24	21	25	19	?	9	12.5

Another type of GNSE was described for the first time in 1971 by Patry[20], whereas a more detailed description was published by Tassinari[21]: epilepsy with electrical status epilepticus during slow sleep (ESES). It is a condition characterized by continuous spikes and waves occurring during at least 85% of the time of slow sleep; during REM-sleep the electrical status epilepticus disappears. Before ESES patients may have motor seizures, typical or atypical absences; psychomotor development may be normal or retarded.

During ESES severe mental deterioration may occur, together with behaviour disturbances.

Recovery is possible, but considerable residual dysfunction has also been reported[22].

Acquired epileptiform aphasia in children (Landau–Kleffner syndrome) is characterized by a progressive language disturbance which is most probably caused by epileptic discharges which occur especially during sleep[23]. The syndrome is probably related to ESES.

Partial status epilepticus

Partial elementary SE is rare in children and is in most cases (75%) somatomotor in expression. When myoclonic jerks persist between the partial seizures, epilepsia partialis continua is present (EPC).

EPC is mainly limited to already neurologically impaired children[24]. Complex partial SE in children is recognized by impairment of consciousness, emotional or behavioral problems, lack of response to familiar persons, lipsmacking, picking at nearby objects and focal clonic activity. EEG investigation is necessary, in order to distinguish it from absence SE. Complex partial SE may also occur in infants (25).

Because of the clinical presentation, various authors discuss complex partial SE together with nonconvulsive SE.

STATUS EPILEPTICUS AMONG CHILDREN IN THE NETHERLANDS

Methods

The co-operation of neurologists in 50 different hospitals in The Netherlands was requested.

Fourteen hospitals agreed to co-operate, but two were unable to provide adequate documentation of admissions and discharges. In addition, data from two of the three epilepsy centres in The Netherlands was included.

We restricted this retrospective study to patients aged between 28 days and 15 years with various types of SE between 1980 and 1987. We visited every hospital and made an on-the-spot investigation of the patient's files.

Only cases with a minimum seizure duration of 30 minutes or an established succession of generalized convulsive seizures without regaining consiousness between the seizures were included.

The patient was given a number to prevent any possibility of recognition and the following data were gathered: age, gender, duration of SE, type of SE, prior epilepsy or not, cause of prior epilepsy, cause of SE, precipitating factors, medical complications, therapy, admission to intensive care unit (ICU), results and outcome. Morbidity was defined as all new neurological signs occurring during the period of SE and was calculated per event.

The causes of morbidity were classified as (a) the underlying cause itself (cause), (b) the seizures themselves and the accompanying medical complications (SE), or (c) cases in which the contribution of the underlying cause or GC–SE itself could not exactly be established (unknown).

The causes of mortality were also classified in this way.

Outcome was described as the neurological condition at the time of hospital discharge.

We obtained from the SIG, the Dutch documentation centre which co-ordinates and collects nationwide hospital statistics, the number of yearly cases of SE and compared these data with ours.

All hospitals which we visited report their data about SE to the SIG. Causes of death however, are registered by another centre, the Central Bureau of Statistics, of the government department of Health Statistics. We asked this centre how many patients die every year as a result of SE.

Results

In The Netherlands, with a population of about 15 million people, according to the SIG every year about 144 children are admitted becausae of SE[26]. Most of these, i.e. 85%, are reported to have had generalized convulsive SE. During our study of SE several hospitals in The Netherlands we found 112 children. One third had been admitted to the ICU (Table 2).

The greater part (67%) were known to have suffered prior epilepsy. About half of the total group (54%) were mentally retarded. Of these,

Table 2: Main characteristics of 112 children with status epilepticus. When admission to the Intensive Care was necessary the patient is categorized IC; if not as ward. Epilepsy and mental retardation refer to the situation before the occurrence of SE

Age	6 months–1 year		1–5 years		5–15 years	
	Ward	IC	Ward	IC	Ward	IC
Male (54)	0	5	8	9	27	5
Female (58)	2	2	13	10	25	6
Epilepsy yes (75)	1	0	17	9	40	8
no (37)	1	7	4	10	12	3
Mental retardation yes (60)	0	0	15	8	33	4
no (52)	2	7	6	11	19	7
Convulsive SE (82)	2	7	18	19	27	9
Nonconvulsive SE (27)	0	0	3	0	22	2
Partial Elementary SE (3)	0	0	0	0	3	0
Causes						
Prior epilepsy (75)						
Acute Symptomatic	0	0	0	0	0	1
Medication problems	0	0	0	2	8	0
Systemic infections	0	0	9	6	1	2
Unknown	1	0	7	1	27	3
Progressive neurol. disease	0	0	0	1	5	1
No prior epilepsy (37)						
Acute symptomatic	0	6	1	9	6	3
Remote symptomatic/ Chronic encephalopathy	0	0	0	0	2	0
Unknown	0	0	1	0	3	1
Febrile	1	1	3	0	0	0

only two had never had seizures before, and both presented with GC-SE.

Generalized convulsive SE

Most patients had generalized convulsive SE (82), among them one patient with tonic and three with myoclonic SE. Generalized convulsive SE was strikingly asymmetrical in 33 patients.

The number of patients with prior epilepsy was 51; 41 were known to suffer mental retardation.

In patients with prior epilepsy a progressive neurological disease was considered responsible for convulsive SE in five cases: Alpers disease (1), neuronal ceroidlipofucsinosis (1), mitochondrial encephalomyopathy (2), unknown cause (1).

The cause remained unknown in 23 patients with convulsive SE. Precipitating factors were systemic infection in 16 cases (13 patients younger than five years) problems with antiepileptic drugs in five patients and in one case stress. In one patient a growing porencephalic cyst caused SE.

In 31 cases without prior epilepsy we found the following causes: acute symptomatic causes were present in 26 cases and considered of viral encephalitis (herpes simplex in three, varicella in one, virus unknown in four), bacterial meningitis (pneumococcus in two), hypoxia (caused by respiratory infection in four), Rye-syndrome (1), metabolic disturbance (dehydration in one), toxic cause (CO in one, cytostatic drugs in one, carbamazepine in one, measles vaccination in one), cerebral contusion (4) and a space occupying lesion due to a ruptured AV-anomaly (1). In five cases febrile SE was present.

Nonconvulsive SE (NSE)

NSE consisted of ESES in four patients, typical absence SE in only two (both initial), atypical absence SE in six and complex partial SE in 15 patients. This group of 15 patients may appear somewhat large: in three the distinction with atypical absence was difficult, an EEG was not performed, all three were mentally retarded. The clinical presentation, however, was suggestive. In another eight patients the clinical distinction was obvious; in the remaining four, clinical presentation and EEG diagnosis were in accordance with the diagnosis of complex partial SE.

The two patients with typical absence SE were a boy of 11 years and a girl of 9 years. The cause of SE remained unknown.

All cases with atypical absence SE had previously been diagnosed with epilepsy and mental retardation; five were between 5 and 15 years of age, only one was between 1 and 5 years. The cause or precipitating factors remained unknown.

Most patients with complex partial SE (12) were known to have suffered prior epilepsy and eight with mental retardation. In cases without prior epilepsy (3) viral meningitis caused SE in one patient; in another patient a remote symptomatic cause was present (cerebral infarction); a systemic infection with fever was present in the third patient. Precipitating factors included medication problems in five. The three patients with elementary partial SE (EPSE) were five, eight and 12 years of age; two were known to have epilepsy and a progressive neurological disorder of unknown origin. The third patient had EPSE because of a head trauma and never had seizures before.

Duration

Duration of convulsive SE did not differ much in patients with or without pre-existing epilepsy (Table 3).

In five cases a succession of generalized convulsive seizures was present, without regaining consciousness, the exact duration was not documented.

In NSE (excluding cases with ESES) cases with a longer duration were more prevalent in patients with prior epilepsy in comparison to cases without prior epilepsy.

Duration of ESES was more than two years in all four cases, duration of partial elementary SE was weeks in two and 21 months in one patient.

Duration and outcome

Duration and convulsive SE appears very variable; the shorter the duration, the less the number of cases with sequelae or death.

Those with a brief duration of convulsive SE (30–60 minutes) account for 18% of morbidity (two cases) and 9% of mortality (one cases). If the duration is between 2 and 8 hours 27% of morbidity and 18% of mortality is accounted for. At a duration of 8 hours half of the morbidity and 27% of mortality is present.

If one considers only cases in which the cause of morbidity or mortality can be directly attributed to the SE and not to the underlying disease, then there is no morbidity prior to two hours duration and only one patient died. Again, only after more than 8 hours duration over half of the morbidity and mortality cases due to SE are encountered.

Table 3: Duration of SE in cases without and with prior epilepsy. A distinction has been made between generalized convulsive SE (GC–SE) and nonconvulsive SE (NSE), which includes complex partial SE. Absolute numbers. Excluded are cases with EPSE (3), ESES (4) and five cases with inadequate documentation of seizure duration

Duration SE	No prior epilepsy (35)		Prior epilepsy (65)	
	GC–SE	NSE	GC–SE	NSE
<2 hours	17	3	26	5
2–8 hours	7	2	9	4
>8 hours	6	0	12	9

Cause and outcome

Overall outcome is worse in patients with an acute symptomatic cause (Table 4). Acute symptomatic causes were more prevalent in children admitted to the Intensive Care Unit (ICU, 60%) in comparison to those admitted to a regular ward (13%). Morbidity (10 out of 11) and mortality (all) occurred mainly in children admitted to the ICU. The cause associated with the highest mortality was anoxia (all four died); in meningoencephalitis morbidity was 40%, no one died.

All cases with febrile SE or SE caused by head trauma had good outcome. Cases with a metabolic cause or space occupying lesion had either morbidity or died, but the number of cases was low.

New neurological signs occurring during or after convulsive SE are presented in Table 5. In nonconvulsive SE mental deterioration was obvious in two cases with ESES. One patient developed complex partial status with a duration of hours because of a viral meningitis and complained of wordfinding problems which resolved in a period of 2 years. Whether this was due to the epileptic discharges or to a encephalitic lesion remains unknown. This patient was not known to have prior epilepsy.

Medical complications and outcome

Medical complications included respiratory insufficience, hyperthermia, acidosis, hypotension, intracranial hypertension, renal and hepatic failure. Outcome is worse in patients with 1 or more medical complications (Table 6). Especially deleterious complications in children were respiratory insufficience and intracranial hypertension. Medical complications occurred almost

Table 4: Outcome of SE in children in relation to type of SE and cause. Partial elementary SE was excluded. Relative numbers. Absolute numbers between brackets

	Good	Morbidity			Mortality		
		SE	Cause	Unknown	SE	Cause	Unknown
Convulsive SE							
Acute symptomatic (31)	55	6.5	3.2	16.1	3.2	9.8	6.5
Pre-existing epilepsy							
with mental retard. (41)	85	2.4	4.9	0	7.3	0	0
without mental retard. (10)	80	0	0	0	10	10	0
Nonconvulsive SE							
No prior epilepsy (5)	80	0	0	20	0	0	0
Prior epilepsy							
with MR (14)	85.7	14.3	0	0	0	0	0
without MR (8)	100	0	0	0	0	0	0

exclusively in convulsive SE, i.e. in 25 patients. The remaining two patients suffered from complex partial SE and in both outcome was good.

In six children with generalized convulsive SE ICP was monitored; in one patient the results were normal. In five children, however, severe intracranial hypertension was present: three patients died (two because of SE, one because of underlying cause), two patients had new neurological problems. In two children the rise of the intracranial pressure was preceded by the start of an electro-encephalographic seizure.

Therapy and outcome

During our investigation we tried to establish the quality of the therapy of SE in relation to outcome. We considered therapy insufficient when an insufficient dose was administered, when the route of administration was wrong (e.g. intramuscular diazepam), when unnecessary delay was present (e.g. waiting for more than 1 hour after diazepam injection, while seizures continued), when mechanical ventilation was not started despite signs of respiratory insufficience or the presence of various medical complications, or when EEG monitoring was not present in cases treated with antiepileptic drugs together with curarisation.

Insufficient therapy in convulsive SE may have contributed to outcome in 13% of the patients, this is not the case in nonconvulsive SE (Table 7).

Table 5: Morbidity in convulsive SE. Absolute numbers

New neurological signs	SE	Cause	Unknown
Persistent myoclonic jerks	0	2	1
Aphasia and blindness	1	0	0
Paresis of the arm	0	1	0
Cognitive problems and hemiparesis	0	0	1
Hemiparesis	1	0	1
Vegetative state	1	0	0
Other	0	0	2
Total	3	3	5

CONCLUSIONS

The incidence of SE in children in The Netherlands is not exactly known; the average annual number reported to the SIG for the period 1980–1987 was for GC–SE 122 (±17), for absence status epilepticus 14 (±5) and for epilepsia partialis continua 8 (±14). The International Classification for Diseases of the World Health Organisation (ICD) does not provide, however, a separate code for complex partial SE, toxic SE or for EPSE with, e.g. aphasia as the sole manifestation. When properly recognized, CPSE may be coded 345.2 (petit-mal SE), but 345.3 (grand-mal) is in our experience also used. Patients with epilepsy and mental retardation who show recurrent periods of SE and live in an asylum or epileptic centre will not always be transferred to a hospital because of SE, because treatment may be successful at their home and the period of SE will not be reported to the SIG.

Patients already hospitalized because of

Table 6: Outcome in children in relation to the presence of medical complications. No complications (no), one complication (1), more than one complication (>1). Relative numbers. Absolute numbers between brackets

	Outcome	Good	Morbidity	Mortality
No	70 (57)	88	9	3
1	12 (10)	80	20	0
>1	18 (15)	13	27	60

Table 7: Quality of therapy in relation to outcome. Relative numbers. Absolute numbers between brackets. Exclusive ESES (4) and EPSE (3)

Outcome	Good	Morbidity	Mortality
Therapy convulsive SE			
Good or sufficient (59)	81.3	8.5	10.2
Insufficient (23)	52.2	26.1	21.7
Therapy nonconvulsive SE			
Good or sufficient (15)	93.3	6.7	0
Insufficient or none (8)	100	0	0

another medical problem who develop SE during their hospital stay present another problem and contribute to the fact that the number of cases reported to the SIG will be too low. Finally a number of cases with simple partial or nonconvulsive SE will not be recognized.

Our number of 112 children with various types of SE will not allow us to estimate the epidemiology of SE more accurately. But the total number and the fact that the cases originated from different types of hospitals (including two epilepsy centres) spread all over the country does permit us to investigate several questions with regard to SE in children, such as cause, therapy and outcome.

The greater part (82) had generalized convulsive SE, especially tonic–clonic SE. We found causes and precipitating factors in agreement to other studies. Our group however showed a higher percentage of prior epilepsy and mental retardation.

The younger age group showed relatively more cases with acute symptomatic causes; the younger the age, the worse the outcome: in children less than 1 year outcome is good in three out of nine (33%), in children 1–5 years in 29 out of 40 (72.5%) and in children 5–15 years in 53 out of 63 (94%). The cause is the main determinant with respect to outcome, not age.

This agrees with the finding that outcome is worse when admitted to the Intensive Care and by the fact that the percentage acute symptomatic causes in patients admitted to the Intensive Care is higher than in patients admitted to a regular ward. Causes associated with poor outcome are tumors, anoxia and metabolic problems[4]. We confirmed this especially with regard to anoxia. The relation between outcome and duration had been investigated also by others[4,8,13,14,16]. In our group we found that outcome in convulsive SE is worse when duration exceeds 8 hours; when we consider only cases with SE as the sole determinant for outcome we found that a duration of more than 2 hours already contributed to a worse outcome. Duration of SE did not depend on the cause; in cases of nonconvulsive SE duration was generally longer.

Outcome in NSE was favourable in most cases; mental deterioration in ESES is part of the syndrome and the wordfindings problems after CPSE in a patient with viral meningitis were temporary. The number of cases with CPSE (15) stresses the fact that CPSE occurs more frequently than acknowledged until now.

The low number of cases with EPSE makes it difficult to draw conclusions. That is why we only mention the main features we found in these three patients. The relation between medical complications and outcome in children has not been reported before in the literature.

Experimental results in animals have stressed the importance of adequate treatment of the medical complications occurring during the course of GC–SE[27,28].

According to the literature in adults the degree of metabolic acidosis did not correlate with outcome, hyperthermia caused a worse outcome[29]. In accordance with the results in 346 adult patients with GCSE[30] we found that outcome in children with convulsive GE is worse with one or more medical complications. Especially intracranial hypertension appeared to be a significant negative factor. In cases with difficult to treat SE and medical complications intracranial pressure should be monitored also because epileptic discharges appeared to cause a rise of intracranial pressure in two of our patients.

A corresponding relation between intracranial pressure and epileptic discharges has been found by others[31,32] and in one of our adult patients. The quality of therapy was questionable in 23 cases of convulsive SE and in eight cases of NSE. Especially in the convulsive cases this may have contributed to outcome. When insufficient, outcome was good in only 12 of the 23 cases (52%), whereas in cases of good therapy outcome was good in 81%. In cases of insufficient therapy morbidity was 26% (in cases of good therapy 8.5%) and mortality 22% (vs. 10.2% in cases of good therapy).

Therapy should be prompt and according to a protocol, including a time schedule, to prevent unnecessary delay. Together with an adequate choice of the antiepileptic drug and prevention of medical complications this may contribute to a more favourable outcome for children with SE[33].

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